UNITED STATES PATENT AND TRADEMARK OFFICE

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ART UNIT	PAPER NUMBER
1656	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

	Application No.	Applicant(s)
	10/812,315	RIEPING, MECHTHILD
Office Action Summary	Examiner	Art Unit	
	ALEXANDER D. KIM	1656	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE _3_ MONTH(S) OR THIRTY (30) DAYS,
WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.
- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed
  after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
  Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any
  earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on _26 February 2009_.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is
    closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) _13-26_ is/are pending in the application.

    4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) _13-26_ is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

    Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

    Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

    a)☐ All b)☐ Some * c)☐ None of:

      1.☐ Certified copies of the priority documents have been received.

      2.☐ Certified copies of the priority documents have been received in Application No. _____.

      3.☐ Copies of the certified copies of the priority documents have been received in this National Stage
    application from the International Bureau (PCT Rule 17.2(a)).

    * See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1)☒ Notice of References Cited (PTO-892)

2)☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)

3)☒ Information Disclosure Statement(s) (PTO/SB/08)
   Paper No(s)/Mail Date _03/04/2009_.

4)☐ Interview Summary (PTO-413)
   Paper No(s)/Mail Date. _____

5)☐ Notice of Informal Patent Application

6)☐ Other: _____.

DETAILED ACTION

Application Status

1.   In response to the previous Office action, a non-Final rejection (mailed on

10/31/2008), Applicants filed a response and amendment received on 02/26/2009. Said

amendment cancelled Claims 1-12; amended Claims 13, 15-16, 21-23.

Claims 13-26 are pending in the instant Office action and will be examined

herein.

It is noted that the instant office action is a non-final Office action in view of at

least one new grounds of rejection.

Information Disclosure Statement

2.   The information disclosure statement (IDS) filed on 03/04/2009 has been

reviewed, and its reference has been considered. A copy of Form PTO/SB/08 is

attached to the instant Office action.

Claim Objections

3.   Claims 13-26 are objected to because of the following informalities:

The use of abbreviation "galP" in Claim 13 (Claims 14-22 dependent therefrom);

abbreviation "PTS" in Claim 23 (Claims 24-26 dependent therefrom). Abbreviations,

unless otherwise obvious and/or commonly used in the art, *e.g.*, "DNA", should not be

recited in the claims without at least once reciting the entire phrase for which the

abbreviation is used in its first appearance in the claims. Appropriate correction is

required.

Withdrawn-Claim Rejections - 35 USC § 112

4.   The previous rejection of Claims 13 (Claims 14-22 dependent therefrom) and 23

(Claims 24-26 dependent therefrom) under of 35 U.S.C. 112, second paragraph, for

Claims 13 (Claims 14-22 dependent therefrom) and Claim 23 (Claims 24-26 dependent

therefrom) reciting limitations "fermenting a bacterium comprising an overexpressed

endogenous DNA sequence encoding the galactose-proton symporter protein in said

bacterium", "said bacterium is an Enterobacteriaceae family" and "said galactose-proton

symporter protein comprises the amino acid sequence of SEQ ID NO: 4 and is encoded

by the nucleotide sequence of SEQ ID NO: 3"; whereas the Enterobacteriaceae

bacterium has to be E. coli because the nucleotide SEQ ID NO: 3 and the polypeptide

of SEQ ID NO: 4 is the E. coli nucleotide or polypeptide (see instant specification page

10, lines 9-11); and as being unclear as to how any Enterobacteriaceae family other

than E. coli can comprise an overexpressed endogenous DNA of SEQ ID NO: 3, or a

DNA encoding SEQ ID NO: 4; is withdrawn by virtue of deleting the term "endogenous".

5.   The previous rejection of Claims 21 and 22 under of 35 U.S.C. 112, second

paragraph, for reciting the limitation "the thrABC operon" or "the tdh gene" wherein the

recitation of "the" in front of the recited operon or gene as referring to the one specific

operon or gene; is withdrawn by virtue of applicant's amendment (i.e., amended to

recite, *e.g.,* "<u>a</u> thrABC operon" and "<u>a</u> tdh gene").

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

5.   Claim 21 is rejected under of 35 U.S.C. 112, second paragraph, as being

indefinite for failing to particularly point out and distinctly claim the subject matter which

applicant regards as the invention.

Claim 21 recites "using a strong promoter, and mutating the ribosome binding

site". With respect to the limitation of "a strong promoter", the scope of those promoters

that are considered to be "strong" is indefinite because the term "strong" is a relative

term and neither the specification nor the claims provide a reference for determining

whether or not the recited promoter is considered to be "strong". Moreover, it is unclear

as to whether or not the limitation encompasses the naturally-occurring promoter,

which, without a reference promoter as noted above, may be considered to be

encompassed by the term "strong promoter".

Also, the limitation "the ribosome binding site" lacks antecedent basis in the claim

because a ribosome binding site is not recited previously. The term "the" refers to a

one particular ribosome binding site; and it is unclear what kind of change (or mutation)

in any particular ribosome binding site would result in overexpression of the gene(s)

recited in Claim 21.

Furthermore, it is noted that the claim requires overexpression of "one or more genes", where "using a strong promoter" and/or "mutating the ribosome binding site" may result in protein overexpression, but would not result in overexpression of one or more genes.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

6.   Claim 21 is rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contain subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a written description rejection.

According to MPEP 2163.II.A.1, in evaluating a claimed invention for adequate written description, the examiner should determine what the claim as a whole covers. "Claim construction is an essential part of the examination process. Each claim must be separately analyzed and given its broadest reasonable interpretation in light of and consistent with the written description. See, e.g., *In re Morris*, 127 F.3d 1048, 1053-54, 44 USPQ2d 1023, 1027 (Fed. Cir. 1997)."

CLAIM INTERPRETATION: Claim 21 limits the genus of microorganisms of the method of claim 13 to further overexpressing one or more of the genes as recited in the claim by (in relevant part) mutating the ribosome binding site, wherein the ribosome

binding site has been interpreted as a site on an mRNA expressed from the

corresponding gene to which a ribosome binds.

MPEP 2163.II.A.2.(a).i) states, "Whether the specification shows that applicant

was in possession of the claimed invention is not a single, simple determination, but

rather is a factual determination reached by considering a number of factors. Factors to

be considered in determining whether there is sufficient evidence of possession include

the level of skill and knowledge in the art, partial structure, physical and/or chemical

properties, functional characteristics alone or coupled with a known or disclosed

correlation between structure and function, and the method of making the claimed

invention".

The specification fails to disclose the reduction to practice of even a single

representative species of the genus of microorganisms with a mutated ribosome binding

site that results in overexpression of one or more of the genes as recited in the claims.

There are no other drawings or structural formulas disclosed of a microorganism with a

mutated ribosome binding site that results in overexpression of one or more of the

genes as recited in the claims. The prior art does not teach any mutated ribosome

binding site for recited genes. Thus, the specification and prior art do not teach any

correlation between structure of any ribosome and function of being able to overexpress

recited protein encoded by the gene. There is no evidence of record that the ribosome

binding site(s) for expressed mRNAs corresponding to the recited genes were known in

the art at the time of the invention. Moreover, there is no teaching or guidance in the

specification or the prior art as to which nucleotide(s) of the mRNA corresponding to the

recited genes could be modified and result in overexpression of the gene. As such, the

specification fails to satisfy the written description requirement of 35 U.S.C. 112, first

paragraph. Because the Claim 21 encompasses very wide structural limitation of any

mutation in any ribosome binding site for recited genes with no functional relationship to

its structural changes in the genus of claim which would results in overexpression of

said gene(s), the one skilled in the art would not be in possession of full scope of the

claimed genus of the instant specification.

Withdrawn-Claim Rejections - 35 USC § 112

7.   The previous rejection of Claim 23 (Claims 24-26 dependent therefrom) under 35

U.S.C. 112, first paragraph, **new matter**, for reciting "transports glucose by a PEP-

dependent phosphotransferase (PTS) pathway", which is not supported by the original

disclosure, is withdrawn by virtue of applicant's amendment.

8.   The previous rejection of Claims 21-22 under 35 U.S.C. § 112, first paragraph,

written description, wherein the breadth of Claim 21 includes a method comprising using

an Enterobacteriaceae family having increased catalytic activity of enzymes from the

thrABC operon according to the disclosure of the term "overexpression" on p. 7, line 1-

5, is withdrawn by virtue of applicant's amendment (i.e., adding the limitation "increasing

copy number" in claim 21; and adding "deleted" in Claim 22.

9.   The previous rejection of Claims 21-22 under 35 U.S.C. § 112, first paragraph,

scope of enablement, because the specification, is withdrawn by virtue of applicant's

amendment (i.e., adding the limitation "increasing copy number" in claim 21; and adding

"deleted" in Claim 22.

Withdrawn-Claim Rejections - 35 USC § 102

10.   The previous rejection of Claim 13-16, 18-20 under 35 U.S.C. 102(b) as being

anticipated by Valle et al. (USPAP 2002/0155521 published on Oct. 24, 2002, as cited

in the previous Office Action) as evidenced by Blattner et al. (1997, Science 277:1453-

1474, as cited in the IDS), is withdrawn by virtue of applicant's amendment (i.e., deleting

the term "operably linking said DNA to a promoter" and adding "changing a promoter

normally found in a galP gene" wherein the reference of Valle et al. did not change a

promoter of the galP gene).

11.   The previous rejection of Claims 13-14 and 17-20 under 35 U.S.C. 102(a) as

being anticipated by Hernandez-Montalvo et al. (2003 Sep. 20, Biotechnol Bioeng, Vol.

83, page 687-694, as cited previously on 12/13/2006) as evidenced by Blattner et al.

(1997, Science 277:1453-1474, as cited in the IDS) and Lee et al. (2003, September,

Journal of Bacteriology, vol. 185, p. 5442-5451, as cited previously on 12/13/2006), is

withdrawn by virtue of applicant's amendment (i.e., reciting the correct coding sequence

of SEQ ID NO: 4 which is residues 33-1427 of SEQ ID NO: 4; thus, it is fully supported

by the instant foreign priority making the instant priority date to 4/1/2003).

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that

form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

12.   Claims 13-20 and 23-24 are rejected under 35 U.S.C. 102(b) as being

anticipated by **Martin et al.** (Biochem. J., 1995, Vol. 308, pages 261-268) as evidenced

by **Roberts** et al. (Journal of Cellular Biochemistry, **1990,** Vol. 44, Issue S14E, on

pages 1-30), "Biosynthesis of amino acids" (prepared by Bryant Miles, April 21, 2003),

QIAGEN-Plasmid Resource Center (QIAGEN product description of Growth of bacterial

cultures, printed 5/12/2009), and Berry et al. (TIBTECH, 1996, Vol. 14, pages 250-256).

Martin et al. teach cloning and overexpression of the galactose-H+ transport

protein, GalP, of Escherichia coli; wherin the E. coli galP gene has 100 % identity to

33-1427 of SEQ ID NO: 3, and the encoded GalP protein has 100% identity to SEQ ID

NO: 4 as evidenced by the sequence alignments below. Martin et al. teach growing the

GalP protein producing E. coli JM1100 containing plasmid pPER3 containing galP

overexpressing the GalP protein (see "Growth of the GalP-producing E. coli strain" on

page 262, top of left column). Martin et al. teach the "yield of GalP in typical preparation

was 40-60% of the inner membrane proteins" (see the end of "Quantification of

overexpressed protein" on page 262, left column). Thus, the method step of Martin et

al. meets the limitations of fermenting an Enterobacteriaceae family overexpressed

DNA sequence encoding the galactose-protein symporter protein (i.e., SEQ ID NO: 4

encoded by the nucleic acid of 33-1472 of SEQ ID NO: 3 which is the E. coli galP gene)

wherein the overexpression is achieved by changing a promoter normally found in a

galP gene. The E. coli of Martin et al. overexpressing GalP protein inherently produces

amino acids (e.g., L-threonine, L-isoleucine, L-valine, L-methionine, L-homoserine and

L-lysine) from a glucose, when the cell were grown in a media, as evidenced by (and it

is well known in the art) the reference of "Biosynthesis of amino acids", which

summarizes the biosynthesis of amino acids and teaches "bacteria can synthesize all

20 of the amino acids" (see bottom of page 1) and "the carbon skeletons comes from

intermediates of glycolysis, the pentose phosphate pathway and the citric acid cycle"

(see middle of page 2) wherein said glycolysis starts with a glucose molecule. Thus,

the amino acid produced in the E. coli of Martin et al. is produced from glucose and the

method of growing E. coli JM1100 overexpressing GalP protein meets all limitations of

Claims 13-16 and 18, wherein an L-amino acid becomes enriched in a fermentation

medium as the total contents of the E. coli (which contains all 20 L-amino acids)

increases. Martin et al. teach isolation of the overexpressed GalP protein by French

press and centrifugation (see middle of left column, page 262); thus, meeting the

limitation of **Claims 19-20.**

The pPER3 vector is derived from pBR322 as evidenced by the reference of

Roberts et al. (Abstract N109 at p. 20, top); wherein the pBR322 has a copy number of

15-20 as evidenced by the reference of Qiagen; thus, the method of Martin et al. meets

the step of increasing copy number of said DNA in **Claim 17.**

The instant claim 23 has additional limitation of said Enterobacteriaceae bacterium comprises PTS enzymes. The instant PTS enzymes in a method of Claim 23 read on any PTS enzymes which exist in E. coli as evidenced by Berry et al. who disclose "The PTS is the major consumer of PEP in E. coli growing on glucose" (see Berry et al., lines 31-32, right column, page 254). Thus, in addition to the reasons above, the E. coli of Martin et al. has many PTS enzymes inherently and meets the limitation of method steps of **Claim 23-24**.

Withdrawn-Claim Rejections - 35 USC § 103

13.   The previous rejection of Claim 21 under 35 U.S.C. 103(a) as being unpatentable over Valle et al. (USPAP 2002/0155521 published on Oct. 24, 2002, as cited in the previous Office Action) in view of Debabov et al. (USP 6,132,999 published on Oct. 17, 2000, as cited in the previous Office Action) is withdrawn by virtue of applicant's amendment overcoming the 35 USC 102 rejection as shown above.

14.   The previous rejection of Claim 22 under 35 U.S.C. 103(a) as being unpatentable over Valle et al. (USPAP 2002/0155521 published on Oct. 24, 2002) in view of Debabov et al. (USP 5,705371 published on Jan. 6, 1998), is withdrawn by virtue of applicant's amendment overcoming the 35 USC 102 rejection as shown above.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all

obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

15.   Claims 13-20 and 23-26 are rejected under 35 U.S.C. 103(a) as being

unpatentable over Debabov et al. (US Patent 5,175,107 issued on Dec. 29, 1992) in

view of Venter et al. (Biochem. J., 2002, Vol. 363, pages 243-252), Flores et al.

(Metabolic Engineering, 2002, Vol. 4, pages 124-137) and Berry et al. (TIBTECH, 1996,

Vol. 14, pages 250-256) and as evidenced by Martin et al. (Biochem. J., 1995, Vol. 308,

pages 261-268), and "Biosynthesis of amino acids" (prepared by Bryant Miles, April 21,

2003).

Debabov et al. (1992) teach teaches a method of making L-threonine using the

E. coli BKIM B-3996 (see Example 1 and 2 in §4-5) containing a recombinant plasmid

pVIC40 (see Abstract). Debabov et al. (1992) teach a method step of growing said E.

coli in a medium having 4% by mass (see bottom of §4) and teach a method of

determining the concentration of the L-threonine as Debabov et al. disclose the

fermentation resulted in a total of 85g/l L-threonine. Debabov et al. also teach many

carbon sources for a fermentation which "Exhibits good growth on saccharose, glucose,

fructose, lactose, mannose, galactose, xylose, glycerol, mannitol" (see §3, lines 52-54).

The difference between Debabov et al. (1992) and the claimed invention is that

the E. coli BKIM B-3996 of Debabov (1992) does not overexpress the E. coli galP gene

(i.e., residues 33-1427 of SEQ ID NO: 3 encoding the SEQ ID NO: 4 protein).

Venter et al. teach the cloning and expression of E. coli GalP (SEQ ID NO: 4)

encoded by the galP gene which were known to be the residues 33-1427 of SEQ ID

NO: 3 as shown in the second (as early as 1980 by Smith et al.) and third (Blattner et al.

1997) sequence alignments at the end of the instant office action.

Flores et al. teach "that GalP can transport, in addition to galactose, other sugars

such as glucose", see left column, lines 14-15, page 125, and further teaches that a

functional galP gene on the chromosome is required for rapid growth on glucose and

teach that it can direct more carbon flux into intermediates or final products of the

aromatic pathway, compared to isogenic PTS$^+$ strains (see left column, lines 18-20,

page 125). Flores acknowledges the concept of metabolic engineering of *E. coli* for the

improvement of industrial strains, including metabolic engineering studies on the main

central metabolic sections that are active during growth on glucose as the sole carbon

source, including overexpression of a gene encoding a protein involved in metabolic flux

(see bottom of left column to right column on p. 124).

Berry et al. teach that influx of Glucose feeds into the glycolysis and the TCA

cycles in Figure 4 on page 254 which produces intermediate compounds that are used

as precursors of all amino acid biosynthesis; which is well known in the art and

summarized by the disclosure of the evidentiary reference of "Biosynthesis of amino

acids", which discloses that "bacteria can synthesize all 20 of the amino acids" (see

bottom of page 1) and "the carbon skeletons comes from intermediates of glycolysis,

the pentose phosphate pathway and the citric acid cycle" (see middle of page 2)

wherein said precursor of amino acid biosynthesis starts with a glucose molecule.

        Therefore, it would have been obvious to one of ordinary skill in the art at the

time the claimed invention was made to practice the method of Debabov et al. (1992)

using the *E. coli* of Debabov et al. overexpressing the galP gene of Venter et al. where

overexpression is achieved by transforming the *E. coli* with a vector encoding galP with

a reasonable expectation of success because the GalP gene increases the carbon flux

into the cell by transporting various sugar including glucose and it can be very effective

transporting the sugar molecules specially when it can be expressed as high as 40 to 60

% of total membrane as shown by Martin et al. (see the end of "Quantification of

overexpressed protein" on page 262, left column); wherein the increase in carbon

source of sugar molecules increases the precursor for the biosynthesis of amino acids

(L-threonine, for example). Debabov et al. (1992) teach the usefulness of L-threonine

which is "known to be an essential amino acid applicable as the component of diverse

nutritive mixture of medical use", "an additive to animals' fodder, as well as reagent for

the pharmaceutical and chemical industries and as a growth factor for microorganisms

producing some other amino acids, such as L-lysine and L-homoserine" (see §1, lines

12-18). Also, Flores et al. teach the PTS system (i.e., PEP:carbohydrate

phoshotransferase system) is the major PEP-utilizing system wherein the PEP is major

precursor of biosynthesis of amino acids (as supported by the glycolysis and the TCA

cycles in Figure 4 on page 254 of Berry et al.) and "the usage of a different system to

internalize and phosphorylate glucose, would lead to an increase in the amount of PEP"

so that PEP would be used in the biosynthesis of many compounds as well as increase

in feeding carbon source into a TCA cycle which in turn increase in amino acid

biosynthesis such as L-threonine. Thus, one skilled in the art would recognize the GalP

transport system as one of said different system as indicated by Flores et al. to

internalize glucose besides the PTS system. One skilled in the art would be motivation

to overexpress the E. coli GalP protein in a L-threonine producing E. coli (BKIM B-3996,

for example) for increase glucose influx in addition to the central glucose influx

PEP:carbohydrate phoshotransferase system (PTS) which would increases overall

carbon source required for amino acids production which is advantageous in an industry

for increasing a cost efficiency which is useful in the amino acid producing industry.

Thus, the claimed invention as a whole was *prima facie* obvious over the combined

teachings of the prior art.

16.   Claim 21 is rejected under 35 U.S.C. 103(a) as being unpatentable over

Debabov et al. (1992) in view of Venter et al., Flores et al., and Berry et al. and as

evidenced by Martin et al., and "Biosynthesis of amino acids" as applied to claims 13-20

and 23-26 above, and further in view of Debabov et al. (USP 6,132,999 published on

Oct. 17, 2000, as cited in the previous non-final office action mailed on 10/31/2008).

        The teachings of Debabov et al. (1992), Venter et al., Flores et al., and Berry et

al. are set forth above. The combination of Debabov et al. (1992), Venter et al., Flores

et al., and Berry et al. does not teach or suggest additionally overexpressing a gene as

recited in claim 21.

Debabov et al. (2000) teach a process of improved amino acid production by
transforming an E. coli with an expression vector comprising a threonine operon
(thrABC), which overexpresses the thrABC gene product. Debabov et al. (2000) teach
a process of making L-threonine by using E. coli BKIIM B-5318 in Example 1. The E.
coli BKIIM B-5318 has "plasmid pPRT614, which has threonine biosynthesis genes
(thrA, B, and C)" as disclosed in the Abstract.

At the time of the invention, it would have been obvious to one of ordinary skill in
the art at the time the claimed invention was made to practice the method of Debabov et
al. (1992) using the *E. coli* of Debabov et al. (1992) overexpressing the galP gene of
Venter et al and further overexpressing the thrABC operon of Debabov et al. (2000),
where overexpression is achieved by transforming the *E. coli* with a vector encoding
galP and thrABC of Debabov et al. (2000) with a reasonable expectation of success
because molecular biology techniques for cloning and expressing multiple genes using
a plasmid vector are well known in the art, and in view of teachings of thrABC operon by
Debabov et al. (1992) and the teaching of E. coli galP gene and its function by Venter et
al. (2002), Flores et al. (2002), Berry et al. (1996). The motivation to do so is provided
by Debabov et al. (2000) et al. who teaches the usefulness of cost-effective and efficient
biosynthetic production of compounds including the L-Thr and that overexpession of
thrABC operon results in enhanced L-Thr production as taught by Debabov et al.
(2000). Thus, the claimed invention as a whole was *prima facie* obvious over the
combined teachings of the prior art.

17.   Claim 22 is rejected under 35 U.S.C. 103(a) as being unpatentable over
Debabov et al. (1992) in view of Venter et al., Flores et al., and Berry et al. and as
evidenced by Martin et al., and "Biosynthesis of amino acids" as applied to claims 13-20
and 23-26 above, and further in view of Debabov et al. (USP 5,705,371 published on
Jan. 6, 1998, as cited in the previous non-final office action mailed on 10/31/2008).

        The teachings of Debabov et al. (1992), Venter et al., Flores et al., and Berry et
al. are set forth above. The combination of Debabov et al. (1992), Venter et al., Flores
et al., and Berry et al. does not teach or suggest deletion of a gene as recited in claim
22.

        Debabov et al. (1998) teach a process of making L-threonine by attenuation of
the tdh gene encoding a threonine dehydrogenase "engaged in degradation of L-
threonine" (see column 2, lines 58-59). Debabov et al. (1998) teach "E. coli strain
VNIIgenetika 472T23" having "insertion of transposon Tn5 into gene tdh " is "devoid
completely of activity" of a threonine dehydrogenase (see column 2, line 53-59).

         At the time of the invention, it would have been obvious to one of ordinary skill in
the art at the time the claimed invention was made to practice the method of Debabov et
al. (1992) using the E. coli of Debabov et al. (1992) overexpressing the galP gene of
Venter et al with a deletion of a tdh gene as taught by Debabov et al. (1998), where
overexpression is achieved by transforming the E. coli with a vector encoding galP with
a reasonable expectation of success because molecular biology techniques for cloning
and expressing multiple genes using a plasmid vector as well as the deletion of known
gene sequences are well known in the art in view of teachings of deletion of tdh gene by

inserting transposon by Debabov et al. (1998) and the teaching of E. coli galP gene and

its function by Venter et al. (2002), Flores et al. (2002), Berry et al. (1996). The

motivation to do so is provided by Debabov et al. (1998) who teach increasing the

production of L-Thr production could be enhanced by deleting tdh gene encoding a

polypeptide that degrades L-Thr. One skilled in the art would be motivation to delete

the tdh gene for usefulness, cost-effective and efficient biosynthetic production of

compounds including the L-Thr by deleting the tdh gene as taught by Debabov et al.

(1998). Thus, the claimed invention as a whole was *prima facie* obvious over the

combined teachings of the prior art.

Conclusion

18.   Any inquiry concerning this communication or earlier communications from the

examiner should be directed to Alexander D. Kim whose telephone number is (571)

272-5266. The examiner can normally be reached on 10AM-6:30PM.

        If attempts to reach the examiner by telephone are unsuccessful, the examiner's

supervisor, Andrew Wang can be reached on (571) 272-0811. The fax phone number

for the organization where this application or proceeding is assigned is 571-273-8300.

        Information regarding the status of an application may be obtained from the

Patent Application Information Retrieval (PAIR) system. Status information for

published applications may be obtained from either Private PAIR or Public PAIR.

Status information for unpublished applications is available through Private PAIR only.

For more information about the PAIR system, see http://pair-direct.uspto.gov. Should

you have questions on access to the Private PAIR system, contact the Electronic

Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a

USPTO Customer Service Representative or access to the automated information

system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Alexander D Kim/
Examiner, Art Unit 1656

Sequence Alignments

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10/812315
RESULT 2
GALP_ECOLI
ID   GALP_ECOLI     STANDARD;      PRT;    464 AA.
AC   P0AEP1; P37021;
DT   20-DEC-2005, integrated into UniProtKB/Swiss-Prot.
DT   20-DEC-2005, sequence version 1.
DT   07-MAR-2006, entry version 5.
DE   Galactose-proton symporter (Galactose transporter).
GN   Name=galP; OrderedLocusNames=b2943;
OS   Escherichia coli.
OC   Bacteria; Proteobacteria; Gammaproteobacteria; Enterobacteriales;
OC   Enterobacteriaceae; Escherichia.
OX   NCBI_TaxID=562;
RN   [1]
RP   NUCLEOTIDE SEQUENCE [GENOMIC DNA], AND CHARACTERIZATION.
RA   Roberts P.E.;
RL   Thesis (1992), University of Cambridge, United Kingdom.
RN   [2]
RP   NUCLEOTIDE SEQUENCE [LARGE SCALE GENOMIC DNA].
RC   STRAIN=K12 / MG1655;
RX   MEDLINE=97426617; PubMed=9278503; DOI=10.1126/science.277.5331.1453;
RA   Blattner F.R., Plunkett G. III, Bloch C.A., Perna N.T., Burland V.,
RA   Riley M., Collado-Vides J., Glasner J.D., Rode C.K., Mayhew G.F.,
RA   Gregor J., Davis N.W., Kirkpatrick H.A., Goeden M.A., Rose D.J.,
RA   Mau B., Shao Y.;
RT   "The complete genome sequence of Escherichia coli K-12.";
RL   Science 277:1453-1474(1997).
RN   [3]
RP   SUBCELLULAR LOCATION.
RC   STRAIN=K12 / MG1655;
RX   PubMed=15919996; DOI=10.1126/science.1109730;
RA   Daley D.O., Rapp M., Granseth E., Melen K., Drew D., von Heijne G.;
RT   "Global topology analysis of the Escherichia coli inner membrane
RT   proteome.";
RL   Science 308:1321-1323(2005).
CC   -!- FUNCTION: Uptake of galactose across the boundary membrane with
CC       the concomitant transport of protons into the cell (symport
CC       system).
CC   -!- SUBCELLULAR LOCATION: Bacterial cell inner membrane; multi-pass
CC       membrane protein.
CC   -!- SIMILARITY: Belongs to the major facilitator superfamily. Sugar
CC       transporter family.
CC   -----------------------------------------------------------------------
CC   Copyrighted by the UniProt Consortium, see http://www.uniprot.org/terms
CC   Distributed under the Creative Commons Attribution-NoDerivs License
CC   -----------------------------------------------------------------------
DR   EMBL; U28377; AAA69110.1; -; Genomic_DNA.
DR   EMBL; U00096; AAC75980.1; -; Genomic_DNA.
DR   PIR; F65079; F65079.
DR   GenomeReviews; U00096_GR; b2943.
DR   EchoBASE; EB2068; -.
DR   EcoGene; EG12148; galP.
DR   BioCyc; EcoCyc:GALP-MONOMER; -.
DR   LinkHub; P37021; -.
DR   PROSITE; PS50850; MFS; 1.
DR   PROSITE; PS00216; SUGAR_TRANSPORT_1; 1.
DR   PROSITE; PS00217; SUGAR_TRANSPORT_2; 1.
KW   Complete proteome; Inner membrane; Membrane; Sugar transport; Symport;
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FT                                   /FTId=PRO_0000050292.
FT    TOPO_DOM      1      15        Cytoplasmic (Potential).
FT    TRANSMEM     16      36        1 (Potential).
FT    TOPO_DOM     37      56        Periplasmic (Potential).
FT    TRANSMEM     57      77        2 (Potential).
FT    TOPO_DOM     78      84        Cytoplasmic (Potential).
FT    TRANSMEM     85     105        3 (Potential).
FT    TOPO_DOM    106     112        Periplasmic (Potential).
FT    TRANSMEM    113     133        4 (Potential).
FT    TOPO_DOM    134     139        Cytoplasmic (Potential).
FT    TRANSMEM    140     160        5 (Potential).
FT    TOPO_DOM    161     171        Periplasmic (Potential).
FT    TRANSMEM    172     192        6 (Potential).
FT    TOPO_DOM    193     250        Cytoplasmic (Potential).
FT    TRANSMEM    251     271        7 (Potential).
FT    TOPO_DOM    272     290        Periplasmic (Potential).
FT    TRANSMEM    291     311        8 (Potential).
FT    TOPO_DOM    312     321        Cytoplasmic (Potential).
FT    TRANSMEM    322     342        9 (Potential).
FT    TOPO_DOM    343     351        Periplasmic (Potential).
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FT    TOPO_DOM    373     394        Cytoplasmic (Potential).
FT    TRANSMEM    395     415        11 (Potential).
FT    TOPO_DOM    416     416        Periplasmic (Potential).
FT    TRANSMEM    417     437        12 (Potential).
FT    TOPO_DOM    438     464        Cytoplasmic (Potential).
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  Query Match              100.0%;  Score 2359;  DB 1;  Length 464;
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  Matches  464;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

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Qy      181 AILLLIGVFFLPDSPRWFAAKRRFVDARVLLRLRDTSAEAKRELDEIRESLQVKQSGWA 240
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Db      181 AILLLIGVFFLPDSPRWFAAKRRFVDARVLLRLRDTSAEAKRELDEIRESLQVKQSGWA 240

Qy      241 LFKENSNFRRAVFLGVLLQVMQQFTGMNVIMYYAPKIFELAGYTNTTEQMWGTVIVGLTN 300
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Qy      301 VLATFIAIGLVDRWGRKPTLTLGFLVMAAGMGVLGTMMHIGIHSPSAQYFAIAMLLMFIV 360
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Qy      361 GFAMSAGPLIWVLCSEIQPLKGRDFGITCSTATNWIANMIVGATFLTMLNTLGNANTFWV 420
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AC   Q2M9Q0;
DT   21-FEB-2006, integrated into UniProtKB/TrEMBL.
DT   21-FEB-2006, sequence version 1.
DT   07-MAR-2006, entry version 2.
DE   D-galactose transporter.
GN   Name=galP;
OS   Escherichia coli W3110.
OC   Bacteria; Proteobacteria; Gammaproteobacteria; Enterobacteriales;
OC   Enterobacteriaceae; Escherichia.
OX   NCBI_TaxID=316407;
RN   [1]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=81053692; PubMed=6159575;
RA   Smith D.R., Calvo J.M.;
RT   "Nucleotide sequence of the E coli gene coding for dihydrofolate
RT   reductase.";
RL   Nucleic Acids Res. 8:2255-2274(1980).
RN   [2]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RA   Sekiguchi T., Ortega-Cesena J., Nosoh Y., Ohashi S., Tsuda K.,
RA   Kanaya S.;
RT   "DNA and amino-acid sequences of 3-isopropylmalate dehydrogenase of
RT   Bacillus coagulans. Comparison with the enzymes of Saccharomyces
RT   cerevisiae and Thermus thermophilus.";
RL   Biochim. Biophys. Acta 867:36-44(1986).
RN   [3]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RA   Chen H., Sun Y., Stark T., Beattie W., Moses R.E.;
RT   "Nucleotide sequence and deletion analysis of the polB gene of
RT   Escherichia coli.";
RL   DNA Cell Biol. 9:613-635(1990).
RN   [4]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RA   Smallshaw J.E., Kelln R.A.;
RT   "Cloning, nucleotide sequence and expression of the Escherichia coli
RT   K-12 pyrH gene encoding UMP kinase.";
RL   Genetics (Life Sci. Adv.) 11:59-65(1992).
RN   [5]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RA   Hayashi K., Morooka N., Yamamoto Y., Fujita K., Isono K., Choi S.,
RA   Ohtsubo E., Baba T., Wanner B.L., Mori H., Horiuchi T.;
RT   "Highly accurate genome sequences of Escherichia coli K-12 strains
RT   MG1655 and W3110.";
RL   Mol. Syst. Biol. 0:0-0(2006).
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RN   [6]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   PubMed=16397293; DOI=10.1093/nar/gkj150;
RA   Riley M., Abe T., Arnaud M.B., Berlyn M.K., Blattner F.R.,
RA   Chaudhuri R.R., Glasner J.D., Horiuchi T., Keseler I.M., Kosuge T.,
RA   Mori H., Perna N.T., Plunkett G. III, Rudd K.E., Serres M.H.,
RA   Thomas G.H., Thomson N.R., Wishart D., Wanner B.L.;
RT   "Escherichia coli K-12: a cooperatively developed annotation snapshot-
RT   -2005.";
RL   Nucleic Acids Res. 34:1-9(2006).
RN   [7]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=97349980; PubMed=9205837; DOI=10.1093/dnares/4.2.91;
RA   Yamamoto Y., Aiba H., Baba T., Hayashi K., Inada T., Isono K.,
RA   Itoh T., Kimura S., Kitagawa M., Makino K., Miki T., Mitsuhashi N.,
RA   Mizobuchi K., Mori H., Nakade S., Nakamura Y., Nashimoto H.,
RA   Oshima H., Oyama S., Saito N., Sampei G., Satoh Y., Sivasundaram S.,
RA   Tagami H., Takahashi H., Takeda J., Takemoto K., Uehara K., Wada C.,
RA   Yamagata S., Horiuchi T.;
RT   "Construction of a contiguous 874-kb sequence of the Escherichia coli
RT   -K12 genome corresponding to 50.0-68.8 min on the linkage map and
RT   analysis of its sequence features.";
RL   DNA Res. 4:91-113(1997).
RN   [8]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=97251358; PubMed=9097040; DOI=10.1093/dnares/3.6.379;
RA   Itoh T., Aiba H., Baba T., Fujita K., Hayashi K., Inada T., Isono K.,
RA   Kasai H., Kimura S., Kitakawa M., Kitagawa M., Makino K., Miki T.,
RA   Mizobuchi K., Mori H., Mori T., Motomura K., Nakade S., Nakamura Y.,
RA   Nashimoto H., Nishio Y., Oshima T., Saito N., Sampei G., Seki Y.,
RA   Sivasundaram S., Tagami H., Takeda J., Takemoto K., Wada C.,
RA   Yamamoto Y., Horiuchi T.;
RT   "A 460-kb DNA sequence of the Escherichia coli K-12 genome
RT   corresponding to the 40.1-50.0 min region on the linkage map.";
RL   DNA Res. 3:379-392(1996).
RN   [9]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=97251357; PubMed=9097039; DOI=10.1093/dnares/3.6.363;
RA   Aiba H., Baba T., Fujita K., Hayashi K., Inada T., Isono K., Itoh T.,
RA   Kasai H., Kashimoto K., Kimura S., Kitakawa M., Kitagawa M.,
RA   Makino K., Miki T., Mizobuchi K., Mori H., Mori T., Motomura K.,
RA   Nakade S., Nakamura Y., Nashimoto H., Nishio Y., Oshima T., Saito N.,
RA   Sampei G., Seki Y., Sivasundaram S., Tagami H., Takeda J.,
RA   Takemoto K., Takeuchi Y., Wada C., Yamamoto Y., Horiuchi T.;
RT   "A 570-kb DNA sequence of the Escherichia coli K-12 genome
RT   corresponding to the 28.0-40.1 min region on the linkage map.";
RL   DNA Res. 3:363-377(1996).
RN   [10]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=97094878; PubMed=8940112; DOI=10.1074/jbc.271.49.31145;
RA   Arn E.A., Abelson J.N.;
RT   "The 2'-5' RNA ligase of Escherichia coli. Purification, cloning, and
RT   genomic disruption.";
RL   J. Biol. Chem. 271:31145-31153(1996).
RN   [11]
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RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=97061202; PubMed=8905232; DOI=10.1093/dnares/3.3.137;
RA   Oshima T., Aiba H., Baba T., Fujita K., Hayashi K., Honjo A.,
RA   Ikemoto K., Inada T., Itoh T., Kajihara H., Kanai K., Kashimoto K.,
RA   Kimura S., Kitagawa M., Makino K., Masuda S., Miki T., Mizobuchi K.,
RA   Mori H., Motomura K., Nakamura Y., Nashimoto H., Nishio Y., Saito N.,
RA   Sampei G., Seki Y., Tagami H., Takemoto K., Wada C., Yamamoto Y.,
RA   Yano M., Horiuchi T.;
RT   "A 718-kb DNA sequence of the Escherichia coli K-12 genome
RT   corresponding to the 12.7-28.0 min region on the linkage map.";
RL   DNA Res. 3:137-155(1996).
RN   [12]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=94261430; PubMed=8202364;
RA   Fujita N., Mori H., Yura T., Ishihama A.;
RT   "Systematic sequencing of the Escherichia coli genome: analysis of the
RT   2.4-4.1 min (110,917-193,643 bp) region.";
RL   Nucleic Acids Res. 22:1637-1639(1994).
RN   [13]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=94240115; PubMed=8183897;
RA   Janosi L., Shimizu I., Kaji A.;
RT   "Ribosome recycling factor (ribosome releasing factor) is essential
RT   for bacterial growth.";
RL   Proc. Natl. Acad. Sci. U.S.A. 91:4249-4253(1994).
RN   [14]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=94124004; PubMed=7904973; DOI=10.1016/0378-1119(93)90470-N;
RA   Allikmets R., Gerrard B.C., Court D., Dean M.C.;
RT   "Cloning and organization of the abc and mdl genes of Escherichia
RT   coli: relationship to eukaryotic multidrug resistance.";
RL   Gene 136:231-236(1993).
RN   [15]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=94018640; PubMed=8412694;
RA   van Heeswijk W.C., Rabenberg M., Westerhoff H.V., Kahn D.D.;
RT   "The genes of the glutamine synthetase adenylylation cascade are not
RT   regulated by nitrogen in Escherichia coli.";
RL   Mol. Microbiol. 9:443-458(1993).
RN   [16]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=93259920; PubMed=8387990;
RA   Zhao S., Sandt C.H., Feulner G., Vlazny D.A., Gray J.A., Hill C.W.;
RT   "Rhs elements of Escherichia coli K-12: complex composites of shared
RT   and unique components that have different evolutionary histories.";
RL   J. Bacteriol. 175:2799-2808(1993).
RN   [17]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=93123180; PubMed=8419307;
RA   Yamada M., Asaoka S., Saier M.H. Jr., Yamada Y.;
RT   "Characterization of the gcd gene from Escherichia coli K-12 W3110 and
RT   regulation of its expression.";
RL   J. Bacteriol. 175:568-571(1993).
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RN   [18]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=93116053; PubMed=1474579;
RA   Cormack R.S., Mackie G.A.;
RT   "Structural requirements for the processing of Escherichia coli 5 S
RT   ribosomal RNA by RNase E in vitro.";
RL   J. Mol. Biol. 228:1078-1090(1992).
RN   [19]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=93094132; PubMed=1459951;
RA   Gervais F.G., Drapeau G.R.;
RT   "Identification, cloning, and characterization of rcsF, a new
RT   regulator gene for exopolysaccharide synthesis that suppresses the
RT   division mutation ftsZ84 in Escherichia coli K-12.";
RL   J. Bacteriol. 174:8016-8022(1992).
RN   [20]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=93077430; PubMed=1447125;
RA   Yamanaka K., Ogura T., Niki H., Hiraga S.;
RT   "Identification and characterization of the smbA gene, a suppressor of
RT   the mukB null mutant of Escherichia coli.";
RL   J. Bacteriol. 174:7517-7526(1992).
RN   [21]
RP   NUCLEOTIDE SEQUENCE.
RC   STRAIN=K-12;
RX   MEDLINE=93011013; PubMed=1396599;
RA   Condon C., Philips J., Fu Z.Y., Squires C., Squires C.L.;

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DB:                   2             Gaps:          0

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Qy     573 GCAATTTTGCTGCTGATTGGTGTCTTCTTCCTGCCAGACAGCCCACGTTGGTTTGCCGCC 632
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Db     181 AlaIleLeuLeuLeuIleGlyValPhePheLeuProAspSerProArgTrpPheAlaAla 200

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Qy     693 GCGAAACGCGAACTGGATGAAATCCGTGAAAGTTTGCAGGTTAAACAGAGTGGCTGGGCG 752
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Qy     993 ACGCTGGGCTTCCTGGTGATGGCTGCTGGCATGGGCGTACTCGGTACAATGATGCATATC 1052
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DEFINITION  Escherichia coli K12 MG1655, complete genome.
ACCESSION   U00096 AE000111-AE000510
VERSION     U00096.2  GI:48994873
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SOURCE      Escherichia coli K12
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  AUTHORS   Blattner,F.R., Plunkett,G., Bloch,C.A., Perna,N.T., Burland,V.,
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            Gregor,J., Davis,N.W., Kirkpatrick,H.A., Goeden,M.A., Rose,D.J.,
            Mau,B. and Shao,Y.
  TITLE     The complete genome sequence of Escherichia coli K-12
  JOURNAL   Science 277 (5331), 1453-1474 (1997)
  PUBMED    9278503
REFERENCE   2  (bases 1 to 4639675)
  AUTHORS   Riley,M., Abe,T., Arnaud,M.B., Berlyn,M.K., Blattner,F.R.,
            Chaudhuri,R.R., Glasner,J.D., Horiuchi,T., Keseler,I.M., Kosuge,T.,
            Mori,H., Perna,N.T., Plunkett,G. III, Rudd,K.E., Serres,M.H.,
            Thomas,G.H., Thomson,N.R., Wishart,D. and Wanner,B.L.
  TITLE     Escherichia coli K-12: a cooperatively developed annotation
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  JOURNAL   (er) Nucleic Acids Res. 34 (1), 1-9 (2006)
  PUBMED    16397293
REFERENCE   3  (bases 1 to 4639675)
  AUTHORS   Arnaud,M., Berlyn,M.K.B., Blattner,F.R., Galperin,M.Y.,
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            Wanner,B.L.
  TITLE     Workshop on Annotation of Escherichia coli K-12
  JOURNAL   Unpublished
  REMARK    Woods Hole, Mass., on 14-18 November 2003 (sequence corrections)
REFERENCE   4  (bases 1 to 4639675)
  AUTHORS   Glasner,J.D., Perna,N.T., Plunkett,G. III, Anderson,B.D.,
            Bockhorst,J., Hu,J.C., Riley,M., Rudd,K.E. and Serres,M.H.
  TITLE     ASAP: Escherichia coli K-12 strain MG1655 version m56
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  JOURNAL    Unpublished
  REMARK     ASAP download 10 June 2004 (annotation updates)
REFERENCE    5  (bases 1 to 4639675)
  AUTHORS    Hayashi,K., Morooka,N., Mori,H. and Horiuchi,T.
  TITLE      A more accurate sequence comparison between genomes of Escherichia
             coli K12 W3110 and MG1655 strains
  JOURNAL    Unpublished
  REMARK     GenBank accessions AG613214 to AG613378 (sequence corrections)
REFERENCE    6  (bases 1 to 4639675)
  AUTHORS    Perna,N.T.
  TITLE      Escherichia coli K-12 MG1655 yqiK-rfaE intergenic region, genomic
             sequence correction
  JOURNAL    Unpublished
  REMARK     GenBank accession AY605712 (sequence corrections)
REFERENCE    7  (bases 1 to 4639675)
  AUTHORS    Rudd,K.E.
  TITLE      A manual approach to accurate translation start site annotation: an
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  JOURNAL    Unpublished
REFERENCE    8  (bases 1 to 4639675)
  AUTHORS    Blattner,F.R. and Plunkett,G. III.
  TITLE      Direct Submission
  JOURNAL    Submitted (16-JAN-1997) Laboratory of Genetics, University of
             Wisconsin, 425G Henry Mall, Madison, WI 53706-1580, USA
REFERENCE    9  (bases 1 to 4639675)
  AUTHORS    Blattner,F.R. and Plunkett,G. III.
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  JOURNAL    Submitted (02-SEP-1997) Laboratory of Genetics, University of
             Wisconsin, 425G Henry Mall, Madison, WI 53706-1580, USA
REFERENCE    10 (bases 1 to 4639675)
  AUTHORS    Plunkett,G. III.
  TITLE      Direct Submission
  JOURNAL    Submitted (13-OCT-1998) Laboratory of Genetics, University of
             Wisconsin, 425G Henry Mall, Madison, WI 53706-1580, USA
REFERENCE    11 (bases 1 to 4639675)
  AUTHORS    Plunkett,G. III.
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  JOURNAL    Submitted (10-JUN-2004) Laboratory of Genetics, University of
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REFERENCE    12 (bases 1 to 4639675)
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ORIGIN
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Disclaimer | Write to the Help Desk
NCBI | NLM | NIH
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Sequences producing significant alignments:                   (Bits)   Value

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gi|882431|gb|U28377.1|ECU28377  Escherichia coli K-12 genome; app  2841   0.0
gi|81244029|gb|CP000036.1|  Shigella boydii Sb227, complete genom  2785   0.0
gi|73854091|gb|CP000038.1|  Shigella sonnei Ss046, complete genom  2769   0.0
gi|81239530|gb|CP000034.1|  Shigella dysenteriae Sd197, complete   2714   0.0
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    D-galactose transporter

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Sbjct 3087881 GGGACGTAAACCAACGCTAACGCTGGGCTTCCTGGTGATGGCTGCTGGCATGGGCGTACT  3087940

Query 1034    CGGTACAATGATGCATATCGGTATTCACTCTCCGTCGGCGCAGTATTTCGCCATCGCCAT  1093
              ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct 3087941 CGGTACAATGATGCATATCGGTATTCACTCTCCGTCGGCGCAGTATTTCGCCATCGCCAT  3088000

Query 1094    GCTGCTGATGTTTATTGTCGGTTTTGCCATGAGTGCCGGTCCGCTGATTTGGGTACTGTG  1153
              ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct 3088001 GCTGCTGATGTTTATTGTCGGTTTTGCCATGAGTGCCGGTCCGCTGATTTGGGTACTGTG  3088060

Query 1154    CTCCGAAATTCAGCCGCTGAAAGGCCGCGATTTTGGCATCACCTGCTCCACTGCCACCAA  1213
              ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct 3088061 CTCCGAAATTCAGCCGCTGAAAGGCCGCGATTTTGGCATCACCTGCTCCACTGCCACCAA  3088120

Query 1214    CTGGATTGCCAACATGATCGTTGGCGCAACGTTCCTGACCATGCTCAACACGCTGGGTAA  1273
              ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct 3088121 CTGGATTGCCAACATGATCGTTGGCGCAACGTTCCTGACCATGCTCAACACGCTGGGTAA  3088180

Query 1274    CGCCAACACCTTCTGGGTGTATGCGGCTCTGAACGTACTGTTTATCCTGCTGACATTGTG  1333
              ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct 3088181 CGCCAACACCTTCTGGGTGTATGCGGCTCTGAACGTACTGTTTATCCTGCTGACATTGTG  3088240

Query 1334    GCTGGTACCGGAAACCAAACACGTTTCGCTGGAACATATTGAACGTAATCTGATGAAAGG  1393
              ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct 3088241 GCTGGTACCGGAAACCAAACACGTTTCGCTGGAACATATTGAACGTAATCTGATGAAAGG  3088300

Query 1394    TCGTAAACTGCGCGAAATAGGCGCTCACGATTAATCTCCCCAAGCTTCCTCCC  1446
              ||||||||||||||||||||||||||||||||||||||||||||||||||||
Sbjct 3088301 TCGTAAACTGCGCGAAATAGGCGCTCACGATTAATCTCCCCAAGCTTCCTCCC  3088353
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